

Exercise duration (in seconds) by exercise test and QOL score assessment by Kansas City Cardiomyopathy Questionnaire (KCCQ) and Serum BNP level. In ivabradine group, patients were started on ivabradine 5 mg in twice daily dose, in addition to OMT. Patients were followed up for 6 months. At the end of six months, LV dimensions, LV function, serum BNP levels, QOL and exercise duration were re-assessed.

Results: At six months, though there was significant reduction of heart rate (70.60 ± 5.06 vs 91.33 ± 8.9 , $p < 0.0001$) and improvement of QOL score ($p = 0.004$) and NYHA functional Class ($p = 0.007$) with ivabradine group compared to OMT group, ivabradine failed to show significant improvement in LVEF (35 ± 3.71 vs 33 ± 4.24 , $p = \text{NS}$), Exercise duration (320 ± 130.6 vs 311.79 ± 103.60 , $p = 0.663$) and BNP level (248.64 ± 175.70 vs 312.57 ± 222.6 , $p = 0.22$). Subgroup analysis showed significant improvement in LVEF (35.71 ± 2.98 vs 33.50 ± 3.73 , $p = 0.003$) in patients with ivabradine who achieved heart rate less than 70 ($n = 25$). No significant adverse effects on ivabradine therapy were noted at the end of six months.

Conclusions: Ivabradine when added to optimal medical therapy, in NYHA Functional Class and QoL in patients with ischemic heart failure. Improvement of Left ventricular function also occurs in presence of adequate heart rate lowering ($\text{HR} < 70/\text{min}$).

Efficacy of levosimendan compared with dobutamine in low-output heart failure

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Background: Levosimendan, a calcium channel sensitiser, improves myocardial contractility without causing an increase in myocardial oxygen demand. This study was done to compare the effects of levosimendan and dobutamine on clinical outcome in patients with low-output heart failure.

Methods: Patients were eligible for participation in this study if they had symptomatic low output heart failure. Overall 175 patients were enrolled in this study. Under continuous haemodynamic monitoring, an initial loading dose of levosimendan of 24 mcg/kg was infused over 10 min, followed by a continuous infusion of 0.1 mcg/kg/min for 24 h. Dobutamine was infused for 24 h at a dose of 5 mcg/kg/min. The primary endpoint was the proportion of patients with clinical improvement.

Results: 100 patients were given levosimendan and 75 dobutamine. The clinical improvement was achieved in 28 (28%) levosimendan-group patients and 15 (20%) in the dobutamine group ($p = 0.02$). At 6 months, 20 (20%) levosimendan-group patients had died, compared with 25 (33%) in the dobutamine group ($p = 0.02$).

Conclusion: In patients with severe, low-output heart failure, levosimendan improved clinical outcome more effectively than dobutamine. Lower mortality was noted in levosimendan group upto 6 months.

Correlation of clinical spectrum, echocardiographic, and angiographic patterns in patients with apical ballooning syndrome in a tertiary care centre of North India

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Background: A cardiac syndrome of “apical ballooning” is being increasingly encountered in routine cardiology practice as it usually mimics acute coronary syndrome at presentation and therefore mandates demonstration of non critical coronary artery disease on coronary angiogram for diagnostic confirmation but its feasibility is scarce in our country. We sought to assimilate clinical, echo-cardiographic and angiographic features of this syndrome at a tertiary care setting from carefully selected cases of apical ballooning and develop an algorithm which should help the emergency physician in making a simpler bedside diagnosis of this syndrome.

Methods: Patients apparently admitted with acute coronary syndrome but subsequently given the diagnosis of transient LV apical ballooning syndrome at our institution from January 2011 to June 2013 were taken prospectively.

Results: Twelve patients were enrolled, mean age was 50 ± 12 years, 10 (83%) were women. Trigger events could be identified in 9 (75%) patients (emotional stress in 3 (25%), post vocal cord surgery in 1 (8%), hemiarthroplasty in 1 (8%), cervical spine surgery in 1 (8%), cervical trauma in 1 (8%), gastrointestinal infection in 1 (8%), road side accident in 1 (8%). Presenting symptoms were; chest pain or discomfort in 3 (25%), NYHA grade III/IV dyspnoea in 9 (75%) patients. 7 (58%) patients had elevated creatine kinase MB and troponin T levels, but the levels were usually only marginally elevated. Electrocardiographic changes observed were ST-segment elevation in 3 (25%), pathological Q waves in 3 (25%), mainly in the leads V_{1-4} . ST-segment depression was found in 4 patients (30%), 3 patients (25%) exhibited T-wave inversion without ST-segment shift. 3 patients presented with cardiogenic shock and 1 patient with ventricular tachycardia. Echocardiographic parameters mean \pm SD LV end-diastolic volume was (115.9 ± 4.0 mL) mean \pm SD LV ejection fraction was (28.2 ± 2.5 %). None of the patients had an E/Em ratio of more than 15. In all 12 patients, left ventricular systolic function recovered completely within three weeks. The systolic strain rate was decreased from base to apex, but the early diastolic strain rate from base to apex was marginally reduced ($+3 \pm 0.5$).

Conclusion: In patients with suspected ABS, clinical history of acute physical/emotional stress with ECG changes mimicking ischemia/infarct, echocardiographic systolic/diastolic paradox with or without contrast echocardiography is helpful in categorization of these patients.

Study of the role of ivabradine in acute heart failure

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Background: Ivabradine is a drug which acts by selectively blocking I_f current in the SA Node. It is approved for use in chronic congestive heart failure. In patients with acute decompensated systolic heart failure, tachycardia could be either a compensatory mechanism or contribute to worsening heart failure. There are situations where using a beta blocker is not an option. The present study was planned to assess the feasibility, safety and efficacy of using Ivabradine in acute heart failure.